British Journal of Obstetrics and Gynaecology
February 1997, Vol. 104, pp. 256–258

SHORT COMMUNICATIONS

The electroencephalogram during normal third trimester pregnancy and six months postpartum

***R. W. M. Keunen** *Neurologist*, ***J. H. R. Vliegen** *Senior Registrar (Neurology)*, ****D. A. E. van der Pol** *Senior Registrar (Gynaecology)*, ****G. Gerretsen** *Obstetrician*, ***C. J. Stam** *Neurologist/Clinical Neurophysiologist*
*Departments of *Neurology and Clinical Neurophysiology and **Obstetrics and Gynaecology, Leyenburg Hospital, The Hague, The Netherlands*

In 1942 Gibbs and Reid described a slowing of the electroencephalogram (EEG) at the end of a normal pregnancy. To the best of our knowledge this is the only report that addresses the modification of the EEG in normal pregnancy. We performed a spectral multichannel EEG analysis and revealed no differences during third trimester pregnancy and six months postpartum. Therefore EEG changes seen during pregnancy, which were previously regarded as 'subtle changes of pregnancy', may turn out to be clinically relevant changes which indicate either pre-existing EEG dysfunction or EEG abnormalities in the context of a pregnancy-related disorder.

Electroencephalophalographic (EEG) changes in healthy individuals, pre- and post delivery, were described by Gibbs and Reid in 1942[1]. They used a three-channel EEG apparatus and recorded both frontal, parietal and occipital areas. Twelve women were studied in the last two weeks of the pregnancy and in the first two weeks postpartum. The authors described 'a slowing of the cortical activity at the end of pregnancy and an absence of high voltage fast records'. It has been cited over and over again, and it lead to a textbook 'knowledge' that the EEG during pregnancy shows 'subtle changes' by the influence of sexual hormones.

In the same article Gibbs and Reid noted that 'slow records occurred as commonly among patients with toxaemia's as among patients with normal pregnancy'. Since then EEG abnormalities in pre- and post-eclampsia have been documented by several authors. Slowing of background activity is observed, especially in the posterior regions, which resolves within six months[2].

Two clinical reasons to perform an EEG during pregnancy are unstable epilepsy and imminent eclampsia. In the case of unstable epilepsy reference EEGs from the patient are usually present. In the case of pre-eclampsia reference EEGs are generally not available. Pre-eclamptic patients often present during the third trimester of pregnancy, and the diagnosis of epilepsy is of utmost importance as the preterm and antenatal eclampsia seems to be particular dangerous to both mother and child[3]. Therefore we conducted a study in healthy normal women, who had an uncomplicated pregnancy. They were studied during their third trimester and six months after delivery.

Methods

Nine women with a mean age of 32 years (range 25 to 40 years) who had an uncomplicated previous history were included in the study. Regular weight, blood and urine testing were within normal limits during their pregnancy. The delivery was uncomplicated in all women. They did not use any drugs during the study period.

EEG studies were performed within the third trimester (week 28 to 36) and after at least six months after delivery (six to eight months). The EEG was recorded with a Cadwell R DC 32 digital EEG apparatus and was stored on an optical disk. From each EEG two representative artefact-free epochs of 2·5 s were chosen during 'eye open' and 'eye closed' state. Technical data regarding the record were: 20 channels; average reference; low pass filter 70 Hz, time-constant 1 s, sample frequency 200 Hz, 8 bit amplitude representation. The epochs were copied as an ASCII file to a personal computer for further analysis. Sixteen channels were included; F8, F7, F4, F3, T4, T3, C4, C3, T6, T5, P4, P3, O2, O1, Fz, Cz, all against the average reference. This reference was

Correspondence: Dr R. W. M. Keunen, Department of Neurology and Clinical Neurophysiology, Leyenburg Hospital, Leyweg 275, 2545 CH The Hague, The Netherlands.

Table 1. Results of ANOVA for different frequency bands. SS = sum of square; MS = mean squares.

	SS	dF	MS	*F*	*P*	ε
α band						
prg	0·046	1	0·046	0·438	0·526	—
Error	0·848	8	0·106			
prg*eye	0·000	1	0·000	0·000	0·998	—
Error	0·413	8	0·052			
prg*side	0·096	15	0·006	0·772	0·706	0·935
Error	0·993	120	0·008			
β band						
prg	0·031	1	0·031	1·589	0·242	—
Error	0·156	8	0·019			
prg*eye	0·024	1	0·024	1·165	0·312	—
Error	0·168	8	0·021			
prg*side	0·044	15	0·003	0·979	0·481	0·574
Error	0·360	120	0·003			
θ band						
prg	0·003	1	0·003	0·120	0·738	—
Error	0·186	8	0·023			
prg*eye	0·001	1	0·001	0·116	0·743	—
Error	0·071	8	0·009			
prg*side	0·024	15	0·002	0·749	0·730	0·610
Error	0·261	120	0·002			
δ band						
prg	0·042	1	0·042	0·418	0·536	—
Error	0·808	8	0·101			
prg*eye	0·016	1	0·016	0·582	0·467	—
Error	0·213	8	0·027			
prg*side	0·108	15	0·007	1·081	0·381	0·543
Error	0·801	120	0·007			

based upon these 16 electrodes. Fp2, Fp1, A2 and A1 were excluded because of frequent muscle artefacts.

Analysis of EEG included a FFT transformation in which the relative power of four different EEG frequency bands was estimated (alpha frequency [α] 8–12 Hz, beta frequency [β] 13–20 Hz, theta frequency [θ] 5–7 Hz and delta frequency [δ] 0–4 Hz). Data were analysed with a three-way repeated-measure ANOVA, using Huynt-Feldt corrected *P* values when appropriate. A separate ANOVA was performed for each of the four dependent variables (relative power in α, β, θ and δ (bands). There were three repeated-measure factors: the pregnant *versus* the nonpregnant condition, the eye open *versus* eye closed state and electrode site (including the 16 leads of the 16 electrodes used). Analysis was done with the statistical package Systat for Windows 5.01. *P* values were set at $< 0{\cdot}05$.

Results

The distribution of the frequency bands for each electrode for all nine women were inspected for each repeated measure factor. These histograms showed a normal distribution on visual inspection. The skewness ranges between –0·30 and +0·30 (mean skewness was 0·197 [SD of the mean 0·140]) indicating a normal distribution of the raw data which justifies the use of ANOVA.

The details of the ANOVA are summarised in Table 1 which includes all the main effects and interactions fitted. The table shows for each frequency band the assessment of whether the frequency band showed differences between the pregnant and the nonpregnant condition (prg), the eye open and eye closed state (prg*eye) and the analysis included an assessment whether the frequency ranges seen at the sixteen different electrode sides is the same during pregnancy as following birth (prg*side).

As seen in the Table ANOVA revealed no significant changes of the EEG during pregnancy and after six month postpartum for any of the frequencies. Neither were differences observed for the prg*eye or prg*side interactions. It means that the significant changes which occur normally in the EEG during eye open and eye closed state are the same during pregnancy and six month postpartum. The same applies for the differences seen between the different electrode sides: pregnancy has no influence on this pattern.

To give an indication that no differences could be found in this study, we present the α frequencies during the eyes closed condition during pregnancy and post delivery. For the occipital electrodes O1 the mean frequencies were respectively 10·14 Hz (SD 0·68) and 10·01 Hz (SD 0·52). For the O2 the frequencies were, respectively, 10·44 Hz (SD 0·95) and 10·14 Hz (SD 0·51). For those familiar with normal EEG readings, highly significant differences were observed for the eye open *versus* eye closed condition especially for the alpha rhythm (dF(1,8); $F = 5461{\cdot}54$; $P < 0{\cdot}001$) (these data are not given in Table 1).

Discussion

The message of this study is a short one. Frequency analysis revealed no significant differences during third trimester pregnancy and six month postpartum in healthy women. This indicates that the EEG is normal in the third trimester pregnancy. One may question the limited number of individuals studied and the short duration of the epoch used in his study. We believe however extending the data would not change the conclusion.

The results are in a way surprising given the earlier observations by Gibbs and Reid[1] and given the impressive endocrinological changes which take place during pregnancy. Up-regulation of the female

hormones, thyroxins and the renine-angiotensine-aldosteron system are known to occur. Moreover, changes in the alpha rhythm have been described during the menstrual cycle, although the hormonal changes which take place during the menstrual cycle are just a fraction of the hormonal changes that take place during pregnancy. Nevertheless, female hormones do not necessarily lead to EEG changes. Harding and Thompson[4] noted that no consistent changes could been found during a period in which oral anticonceptives were used. Thus, although significant changes of female hormones occur during pregnancy, the brain seems to adapt to these changes, given the outcome of the present EEG analysis.

The clinical relevance of this study is that EEG changes seen during pregnancy, which were previously addressed by clinicians as subtle changes of pregnancy may turn out to be clinically relevant changes which indicate either to pre-existing EEG dysfunction or to more recent EEG abnormalities. The observations enhance the significance of interictal EEG abnormalities which can be observed during eclampsia[5]. Moreover, the present findings may be an argument to perform more sophisticated EEG analysis during the third trimester of pregnancy, especially in toxaemic patients, in order to understand the early EEG phenomena which may precede eclampsia.

References

1 Gibbs FA, Reid DE. The electroencephalogram in pregnancy. *Am J Obstet Gynecol* 1942; **44**: 672–675.

2 Donaldson JO. The brain in eclampsia. *Hypertens Preg* 1994; **13**: 115–133.

3 Douglas KA, Redman CGW. Eclampsia in the United Kingdom. *BMJ* 1995; **309**: 1395–1400.

4 Harding GFA, Thompson CRS. EEG rhythms and the internal milieu. In: Remonal A, editor. *Handbook of Electroencephalography and Clinical Neurophysiology*. Amsterdam: Elsevier, 1976: 176–194.

5 Thomas SV, Somananthan N, Radkhakumari R. Interictal EEG changes in eclampsia. *Electroenceph Clin Neurophysiol* 1995; **94**: 271–275.

Received 13 March 1996
Accepted 25 September 1996

British Journal of Obstetrics and Gynaecology
February 1997, Vol. 104, pp. 258–260

Is HTLV-1 status another antenatal screening test that we need?

***C. Otigbah** *Research Registrar*, ***A. Kelly** *Senior House Officer*, ****C. Aitken** *Senior Registrar*,
****J. Norman** *Senior Clinical Scientist*, ****D. Jeffries** *Head of Department of Virology*,
†K. J. Erskine *Consultant Obstetrician and Gynaecologist*

**Homerton Hospital, London; **Department of Virology, The Royal Hospitals Trust, London;*
†The Royal Hospitals Trust and Homerton Hospital, London

Human T-lymphocytic virus type 1 (HTLV-1) carriage is associated with a 2% to 5% lifetime risk of developing a severe, if not fatal, disease. In our Inner London antenatal population, we found that the carrier rate was 0·3%. The antenatal transmission rate is known to be very low, but if the infant is breastfed the transmission rate is up to 25%. It is therefore possible to interrupt the transmission cycle by advising against breastfeeding. The ethical and fiscal issues surrounding antenatal testing are addressed.

Human T-lymphocytic virus type 1 (HTLV-1) carriage is associated with a 2% to 5% lifetime risk of developing leukaemia, lymphoma or tropical spastic paresis. Breastfeeding is one of the major transmission routes, and in Japan a campaign has begun to encourage HTLV-1 carrier mothers not to breastfeed. We investigated our antenatal population in the East End of London and discuss the issues surrounding testing for HTLV-1.

Correspondence: Dr K. J. Erskine, The Royal Hospitals Trust and Homerton Hospital, Hackney, London.

Screening for infectious diseases in pregnancy has always been an accepted part of antenatal care. Standard infection screens include urine examination, serology for syphilis and rubella; more recently, hepatitis B and human immunodeficiency virus (HIV) screening has been offered after counselling. Other infection screens which are not routine in the United Kingdom are those for toxoplasmosis and HTLV-1.

HTLV-1 was the first retrovirus shown to be associated with disease in humans. Data from Japan and the Caribbean suggest that for infected women, there is a risk of adult-T cell leukaemia/lymphoma of